**SUPPORTING INFORMATION**

## 1. Methods

### 1.1. Preparation and characteristics of dscECM

Spinal cord was provided from healthy Sprague–Dawley rats (200–250g of body weight, animal center of Wenzhou medical university). Spinal cord tissues were cut into 15mm segments. Spinal cords were kept quickly at normal saline in the refrigerator (12 h, 4°C). The spinal cord was rinsed in 0.01% phosphate-buffered saline on gyratory shaker (1h, 4°C,60 rpm) (PBS, Solarbio, Beijing), The spinal cord was rocked in 1% Triton X-100 solution for 12h at 4°C to perform decellulization process. And then, the decellulized spinal cord was washed by sterile distilled water for three times (each 10 min), followed by washing with 4.0% deoxycholate for three times at4°C for 8 h. Finally, they were rinsed with 0.01% PBS for ten times. All solutions contained 200 U/ml penicillin (Invitrogen, 15140-122), 200 mg/ml streptomycin (Invitrogen, 15140-122). The samples were stored in 0.01% sterile PBS not more than one week at 4°C.

Decellularization of normal spinal cord was detected according to literature [1]. Briefly, dscECM was fixed in 4% paraformaldehyde, washed with distilled water, dehydrated in graded alcohol, embedded in paraffin and sectioned at 5 μm thickness and stained with hematoxylin and eosin (H&E). All stained sections were analyzed by microscopy.

To confirm the purity of dscECM, the residual nuclear DNA was further detected by gel electrophoresis. Briefly, the homogenized dscECM sample was extracted using E.Z.N.A.® Tissue DNA Kit (OMEGA). 10μl of DNA extract was determined by gel electrophoresis on 1.0% of agarose gel with ethidium bromide (2 h, 60 V) followed by imaging with ultraviolet illumination. The homogenized spinal cord tissue was treated

by smiliar procedure above to extract DNA as control.

**1.2. Analysis of dscECM components**

The immunofluorescence staining of dscECM was firstly analyzed by the method depicted in the literatures. Briefly, paraffin section of dscECM (5μm of thickness) was immunostained for collagen types IV (Col IV), laminin (LM) and fibronectin (FN). The primary antibodies used were the following: polyclonal rabbit anti-mouse collagen type IV, (Abcam; 1:200), polyclonal rabbit anti-mouse fibronectin (Abcam; 1:250) and polyclonal rabbit anti-mouse laminin (Abcam; 1:250) at 4 °C overnight. After extensive washes, the sections were incubated in the appropriate secondary antibodies (Molecular Probes, 1:100) for 1 h, followed by washing with PBS. Species-specific non-immune IgG and omission of primary antibody was used as negative controls. Immunofluorescent staining was analyzed by a Nikon ECLPSE 80i (Nikon, Japan).

The dscECM components, including collagen IV, fibronectin and laminin were further quantified by the ELISA method depicted in the literature [2]. Briefly, the dscECM solution was added to the micro ELISA plate wells and combined to the specific antibody. Then a biotinylated detection antibody specific for Col IV/FN/LN and Avidin-Horseradish Peroxidase (HRP) conjugate is added to each micro plate well for further incubation, respectively. Afterward, the free components are removed and the substrate solution is added to each well for reaction. Then, a sulphuric acid solution was added to terminate the enzyme-substrate reaction. The optical density (OD) is measured at a wavelength of 450 nm. The concentration of Col IV/ FN / LN in dscECM was calculated by comparing the OD of the samples to the standard curve. Periodic Acid-Schiff staining was performed on rehydrated paraffin sections to evaluate the retained glycosaminoglycans (GAGs). GAGs of ECM and native tissue were extracted with 0.1 mg/ml proteinase K (Sigma) in buffer (10 mM Tris–HCl, pH

8.0, for 48 hours at 50°C) at a concentration of 50mg/ml. The sGAG concentration was detected following the manufacturer's recommended protocol of the Blyscan Sulfated Glycosaminoglycan Assay Kit (Biocolor Ltd., UK) (n=3 in duplicate or triplicate).

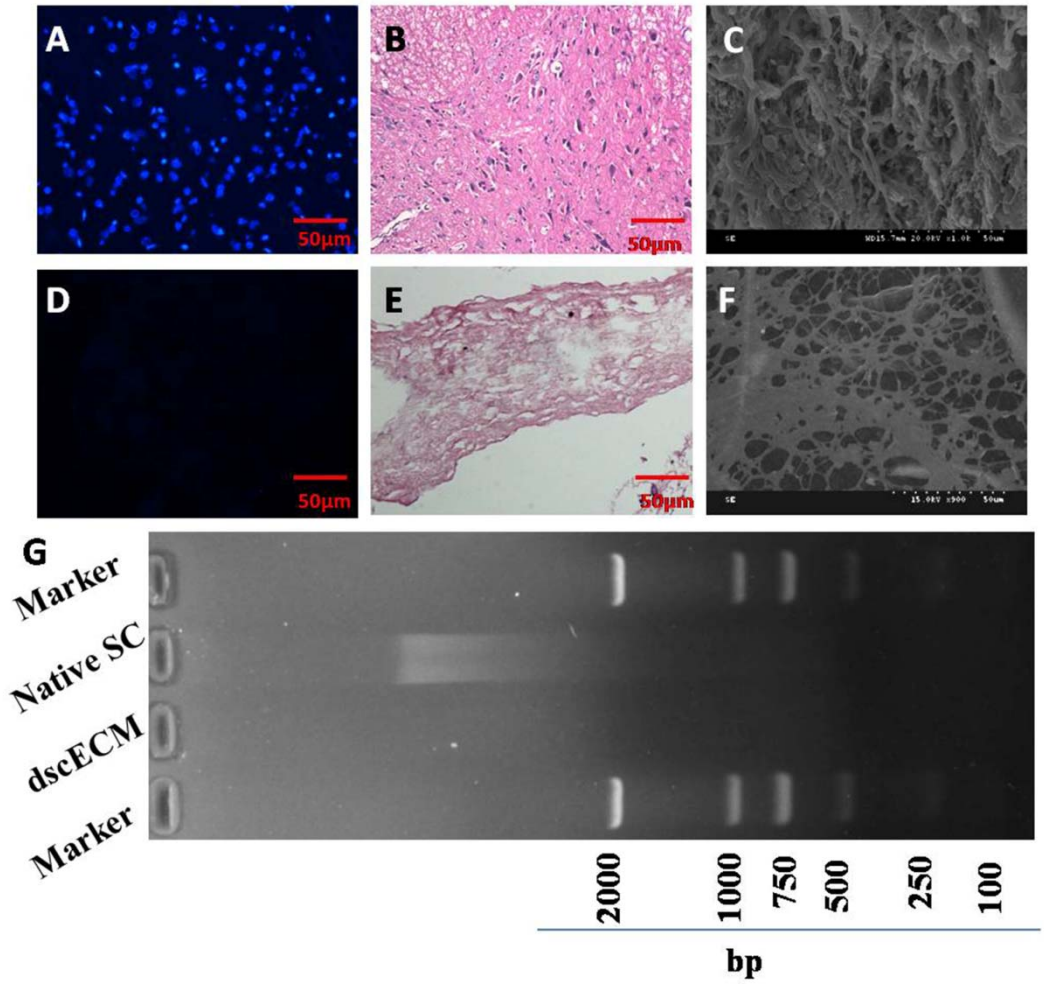


Fig S1. DAPI staining, HE staining and scanning electronic microscopy of native spinal cord (Native SC) (A-C) and dscECM (D-F), respectively; (G) SDS-PAGE electrophoresis of residual nuclear DNA in dscECM and native spinal cord.

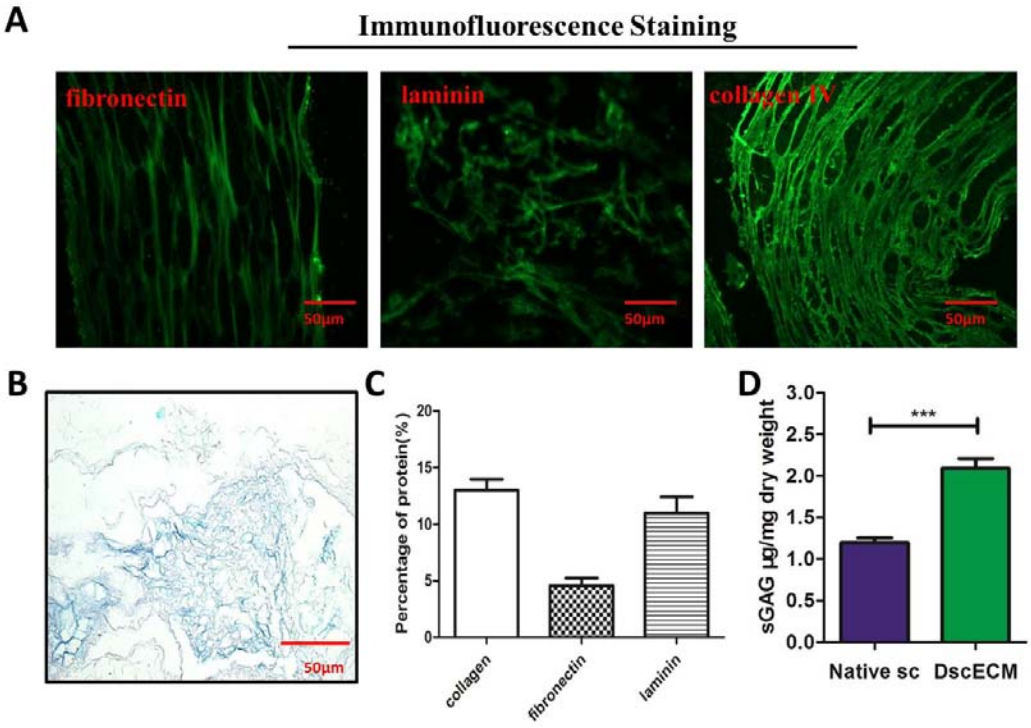


Fig S2. (A) Immunofluorescence staining of protein-typed components of dscECM scaffold; (B) Glycosaminoglycans (sGAG) staining in dscECM by periodic acid-Schiff staining; (C) Quantitative amount of collagen IV, laminin and fibronectin in dscECM scaffolds and (D) Quantitative amount of sGAG analyzed by Blyscan Sulfated Glycosaminoglycan Assay Kit.

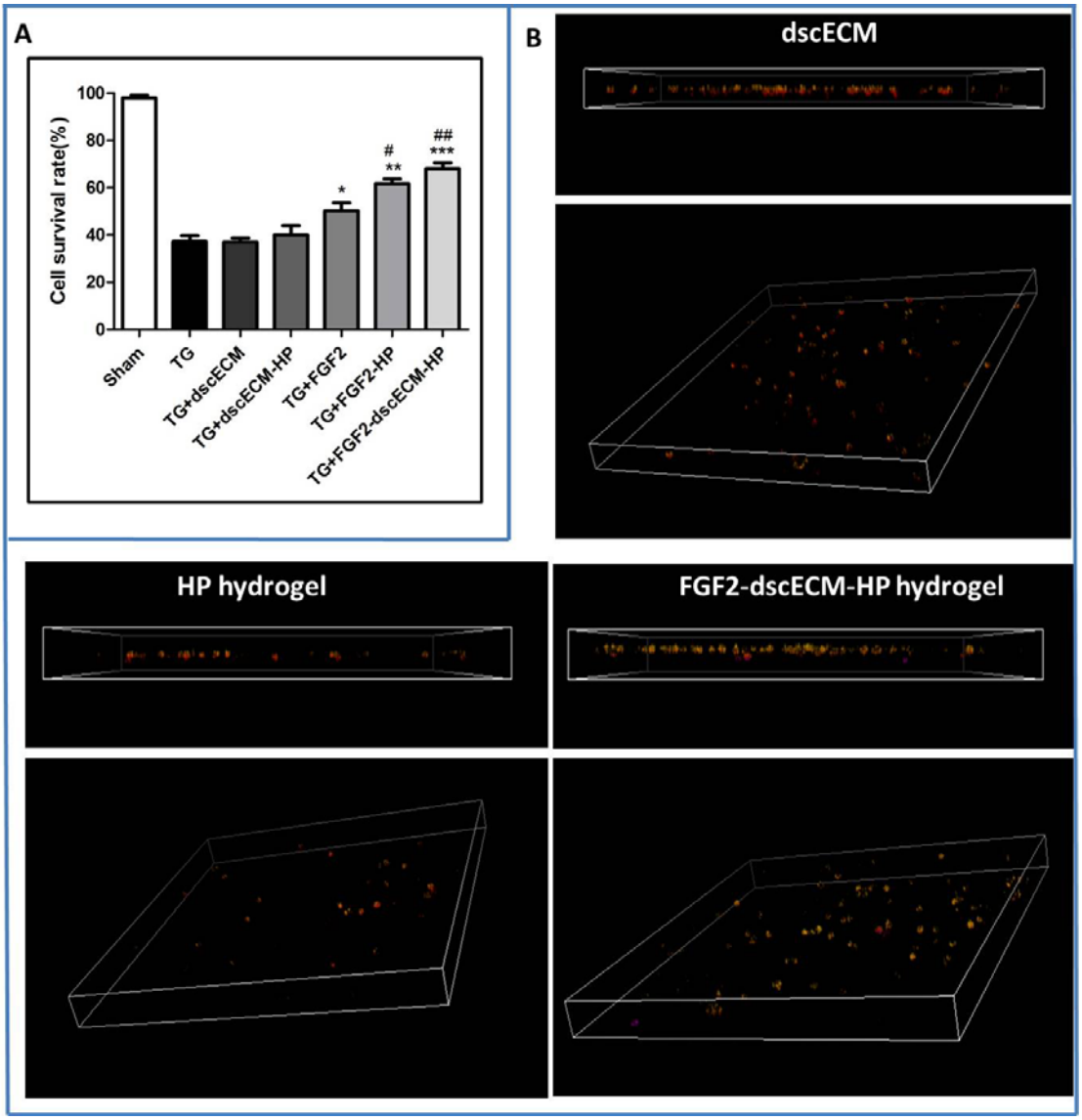


Fig S3. (A) Effects of various treatments on cell proliferation of the thapsigargin-injured PC 12 cells, and (B) The adsorption behavior of PC12 cells on various scaffolds were photographed by confocal laser scanning microscope (Red-fire fluorescence). Data are presented as Mean ± SEM, n=5. *: FGF2-dscECM-HP group VS SCI group, #: FGF2-dscECM-HP group VS FGF2 group, *$P$<0.05, **$P$ < 0.01, ***$P$ < 0.001, #$P$ < 0.05 and ##$P$ < 0.01.

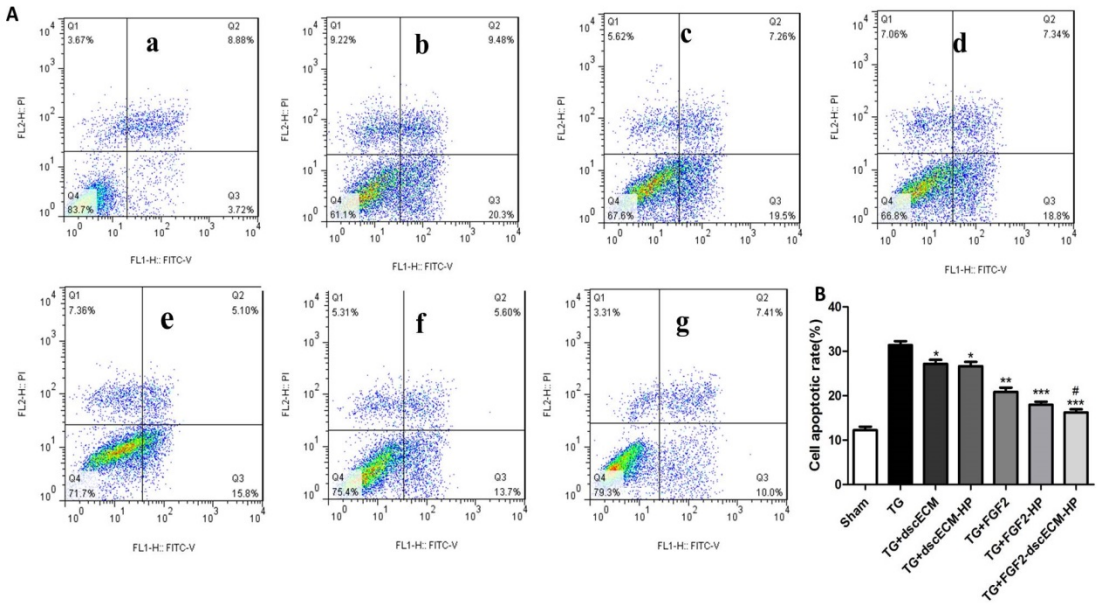


Fig S 4. (A) Apoptosis of PC12 cells analyzed by flow cytometery after different treatment (a: Sham group; b: TG group; c: TG+dscECM group; d: TG+dscECM-HP group e: TG+FGF2 solution group; f: TG+FGF2-HP group g: TG+FGF2-dscECM-HP hydrogel group), and cell apoptotic rate was quantified according to flow cytometery analysis. *: groups VS TG group, #: groups VS FGF2 group, $*P<0.05$, $**P < 0.01$, $***P < 0.001$, $\#P < 0.05$ and $\#\#P < 0.01$.

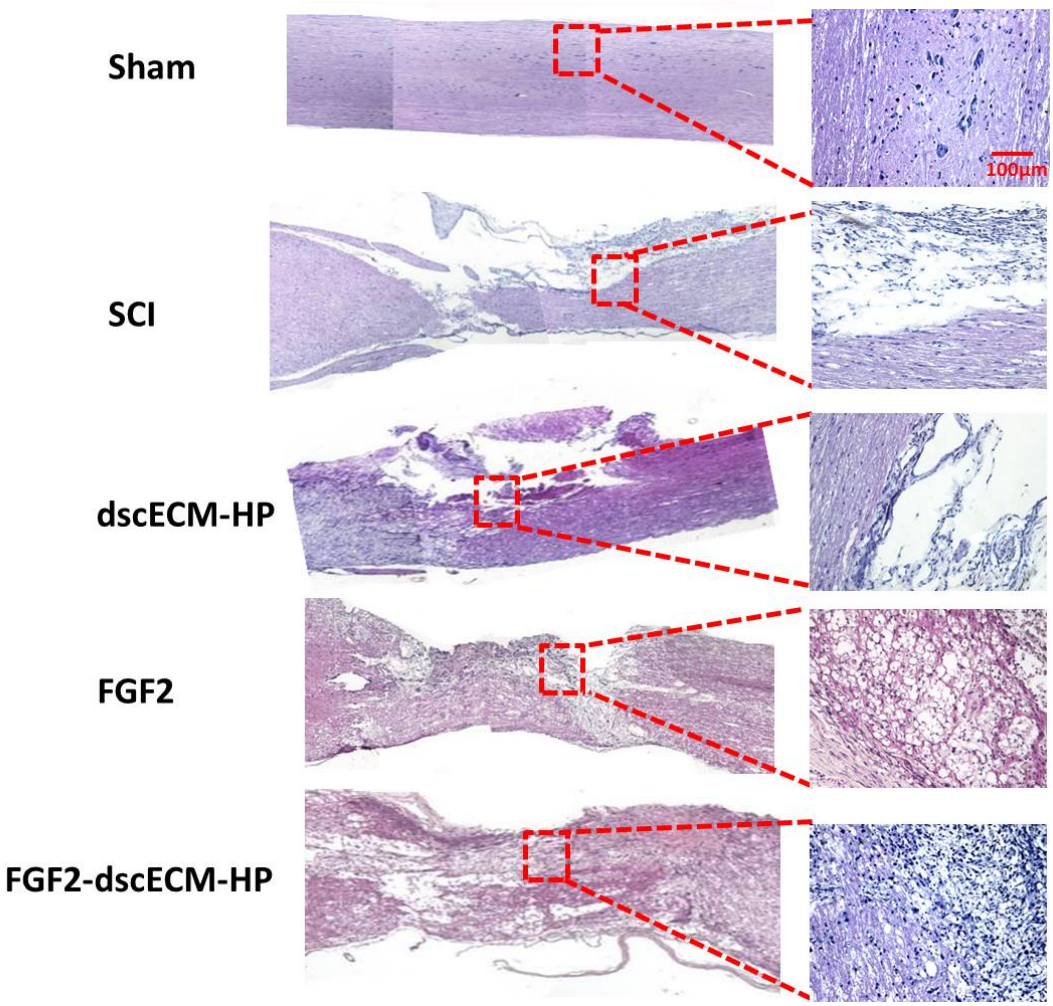


Fig S5. Histopathological HE staining of spinal cord at the junction of injured spinal cord and normal spinal cord on the 28$^{th}$ day after various treatments.

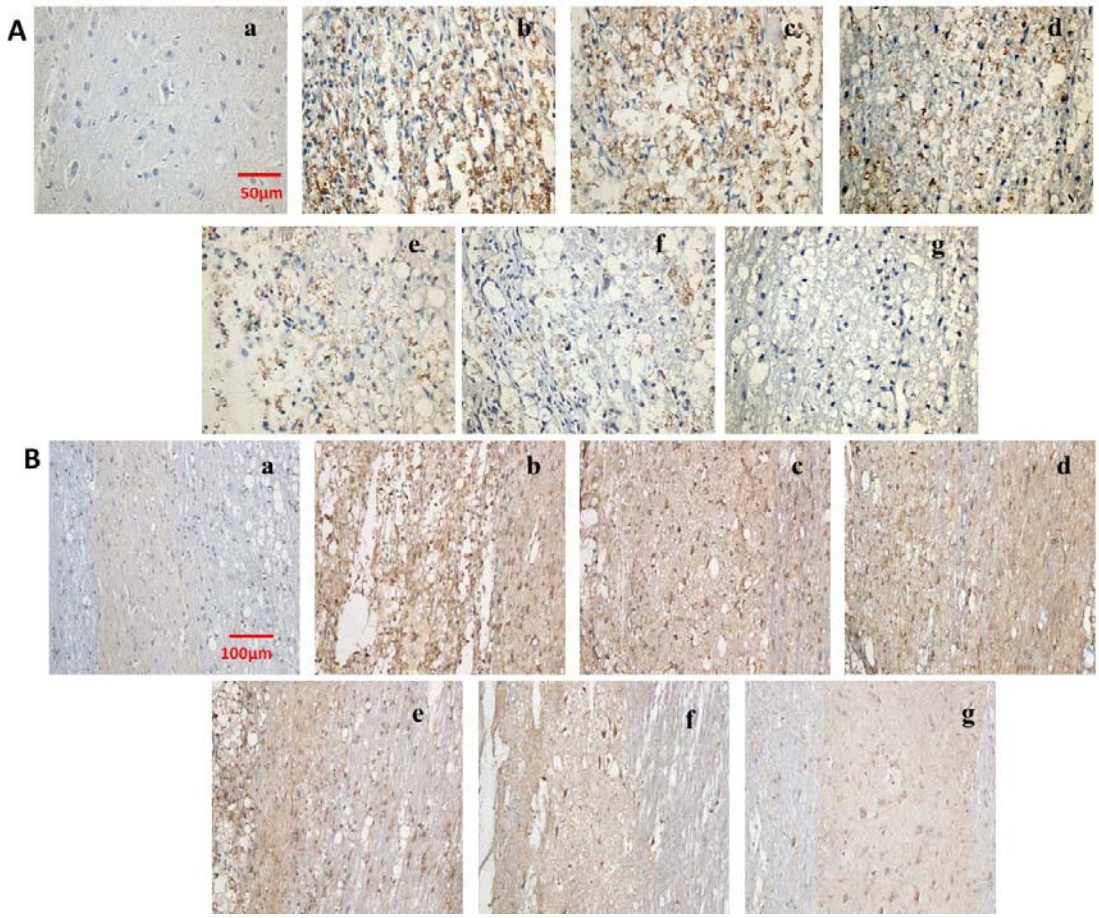


Fig S6. Apoptosis results of SCI rats in vivo after different treatment: Immunohistochemical staining of Caspase-3 (A) (×400) and CHOP (B) (×200) (a: Sham group; b: SCI group; c: ECM group; d: ECM+HP group e: FGF2 solution group; f: FGF2-HP hydrogel group g: FGF2-ECM-HP hydrogel group).

**References**

[1] P.M. Crapo, C.J. Medberry, J.E. Reing, S. Tottey, Y. van der Merwe, K.E. Jones, S.F. Badylak, Biologic scaffolds composed of central nervous system extracellular matrix, Biomaterials 33(13) (2012) 3539-47.
[2] C.J. Medberry, P.M. Crapo, B.F. Siu, C.A. Carruthers, M.T. Wolf, S.P. Nagarkar, V. Agrawal, K.E. Jones, J. Kelly, S.A. Johnson, S.S. Velankar, S.C. Watkins, M. Modo, S.F. Badylak, Hydrogels derived from central nervous system extracellular matrix, Biomaterials 34(4) (2013) 1033-40.